(r = −0.273, p = 0.044). Multivariate analysis confirmed that urinary COL1A1 mRNA level is an independent predictor of serum creatinine doubling (adjusted hazard ratio 1.023, p = 0.016).
Conclusion: Urinary COL1A1 mRNA level is elevated in nephrotic patients irrespective to the pathological diagnosis, and it correlates with proteinuria, histological scarring, and inversely with renal function. Furthermore, urinary COL1A1 mRNA level predicts renal function loss during follow up. Our results suggest that urinary COL1A1 mRNA level may be used for risk stratification of adult nephrotic syndrome.

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0006
Podocyte mRNA in the Urinary Sediment of Minimal Change Nephropathy and Focal Segmental Glomerulosclerosis

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Background: Podocyte depletion is a characteristic feature of progressive renal failure. We hypothesize that studying the podocyte mRNA level in urinary sediment may provide diagnostic and prognostic information in adult nephrotic syndrome.
Methods: We studied 25 patients with minimal change nephropathy (MCN), 25 with focal segmental glomerulosclerosis (FSGS), and 17 healthy controls. The mRNA levels of nephrin, podocin and synaptopodin in urinary sediment were quantified.
Results: There were significant differences in the urinary sediment nephrin and podocin, but not synaptopodin, mRNA levels between diagnosis groups. Post hoc analysis further shows that urinary nephrin mRNA levels of the MCN group are lower than those in the control and FSGS groups, although the difference between MCN and FSGS Groups does not reach statistical significance. The degree of proteinuria inversely correlates with urinary nephrin mRNA levels in the MCN (r = −0.526, p = 0.007) as well as FSGS group (r = −0.521, p = 0.008). For the FSGS group, the rate of renal function decline significantly correlates with baseline urinary synaptopodin mRNA levels (r = −0.496, p = 0.012).
Conclusion: Urinary nephrin and podocin mRNA levels are reduced in patients with MCN and probably FSGS, and the magnitude of reduction correlates with the degree of proteinuria. Urinary synaptopodin mRNA levels correlate with the subsequent rate of renal function decline in patients with FSGS. Our result indicates that urine sediment podocyte mRNA levels provide novel insights in the pathophysiology of nephrotic syndrome and could be useful for risk stratification.

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0008
EGFR Inhibition Alleviates Hyperuricemic Nephropathy in Rats

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Hyperuricemia is an independent risk factor for chronic kidney disease and contributes to kidney fibrosis. In this study, we investigated the effect of epidermal growth factor receptor (EGFR) inhibition on the development of hyperuricemic nephropathy (HN) and the mechanisms involved. In a rat model of HN induced by feeding a mixture of adenine and potassium oxonate, increased EGFR phosphorylation and severe glomerular sclerosis and renal interstitial fibrosis were evident, accompanied by renal dysfunction and increased urine microalbumin excretion. Administration of gefitinib, a highly selective EGFR inhibitor, prevented renal dysfunction, reduced urine microalbumin and inhibited activation of renal interstitial fibroblasts and expression of extracellular proteins. Gefitinib treatment also inhibited hyperuricemia-induced activation of the transforming growth factor-β1 (TGF-β1) and nuclear factor-κB (NF-κB) signaling pathways and expression of multiple profibrogenic cytokines/chemokines in the kidney. Furthermore, gefitinib treatment suppressed xanthine oxidase activity which mediates uric acid production, and preserved expression of organic anion transporters 1 and 3, which promotes uric acid excretion in the kidney of hyperuricemic rats. Thus, blocking EGFR can attenuate development of HN via suppression of TGF-β1 signaling and inflammation, and promotion of the molecular processes that reduce uric acid accumulation in the body.

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0015
Renoprotection by Helix B Surface Peptide in Puromycin Aminonucleoside-induced Nephrotic Syndrome

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Objective: Glial cell line-derived neurotrophic factor (GDNF), a memberof TGF-β superfamily, was testified to have protective effect on podocyte in vitro. Helix B surface peptide (HBSP), derived from erythropoietin, displays powerful tissue protection during kidney ischemia reperfusion (IR) injury without erythropoietic side effects. The purpose of this study was to explore the renoprotection effect of HBSP in puromycin aminonucleoside-induced nephrotic syndrome for the first time, and the change of GDNF involved in.
Methods: Wistar rats were randomly divided into three groups, (1) Control group (n = 4); (2) PAN+vehicle group (n = 6); (3) PAN+HBSP group (n = 8), which treated with HBSP (8 nmol/kg), 4 hours before the injection of PAN (60 mg/kg) and every 24 hours after. Biochemical parameters, gene expression and histology were assessed at 7 days.
Results: The levels of proteinuria significantly increased in rats with PAN-induced nephrosis. Treatment with HBSP significantly prevented these deteriorations induced by PAN. Glomerular lesions, especially vacuolation of podocytes, and the increase of desmin expression in PAN-treated rats were significantly ameliorated by treatment with HBSP. The GDNF was increased in in rats with PAN-induced nephrosis and increased further after treated with HBSP. We also found activation of the RAS/ERK signaling pathway in HBSP-treated rats.
Conclusion: HBSP could ameliorate proteinuria in PAN-induced nephrosis, which might be due to the amelioration of podocyte injury. HBSP inhibited the depletion of podocin. Increasing expression of GDNF and activation of downstream RAS/ERK signaling pathway can be involved in the renoprotective effect of HBSP. GDNF might be involved in the renoprotective effect of HBSP.